

Critical Thinking in Diagnosing Primary Open-angle Glaucoma: A Teaching Case Report

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Abstract

This paper will discuss the care of a patient with glaucoma over a period of time and how critical analysis influences the patient's management. Glaucoma and ocular hypertension are common ocular conditions encountered by many eye care professionals. Primary open-angle glaucoma is the leading cause of irreversible blindness worldwide. Important critical thinking concepts, such as determining appropriate and relevant information, analyzing data, justifying a diagnostic conclusion, evaluating implications, and assessing the role of assumptions and point of view, are used in the analysis and discussion of the case. This case demonstrates the ambiguities and challenges of diagnosis, along with the use of evidence-based medicine, critical thinking, and the art of questioning.

Key Words: glaucoma, critical thinking, ocular hypertension

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Background

This case involves a patient who was initially diagnosed with glaucoma in 2001 and then follows the patient over an 8-year period. During the course of the patient's care, the diagnosis and treatment changed, highlighting how changes in information can alter the diagnosis and treatment of a chronic condition. During the course of the patient's treatment, several vital questions arose: Is this glaucoma? Is this ocular hypertension? Does she need treatment? This case can be analyzed and discussed at a multitude of levels and time frames. It is most appropriate for use with first- and second-year students, who possess a basic knowledge base about glaucoma. At more advanced levels, the case can be used as a review of knowledge and to initiate debate.

Throughout the continuum of learning from first to fourth year, the expectations for a case discussion should change. At the early stages of education, the expectations should focus on how changes in information influence decision-making, development and appreciation of critical thinking concepts, and raising fundamental questions needed to analyze and evaluate a case. As students advance, in addition to the above concepts, the students should be able to independently apply critical thinking concepts, evidence-based medicine and their own clinical experience in the analysis of the case. Students at all levels should be encouraged to identify assumptions, analyze points of view, and recognize the impact of these concepts on the case and a patient encounter.

Primary open-angle glaucoma is the leading cause of irreversible blindness worldwide.¹ In 2004, the overall prevalence of diagnosed open-angle glaucoma in the United States was 2.22 million people or 1.9% of the population over age 40.² It is estimated that an additional 2 million people are undiagnosed.³ By the year 2020, 3.6 million people in the United States will be affected by the disease.¹

In the United States, optometrists are responsible for the detection, diagnosis, and management of glaucoma. This includes identifying patients at risk, assessing risk factors, implementing treatment, and educating the patient to ensure patient adherence and compliance.

The increasing prevalence of primary open-angle glaucoma and the negative consequences of this disease make this case educationally and clinically relevant. Additionally, the increase in scope of practice and responsibility within the profession of optometry along with the challenges associated with the diagnosis of glaucoma emphasize the importance of this case in optometric education. The value of this case does not lie in its uniqueness or rarity but rather in its commonality, prevalence, and ability to initiate debate.

Student Discussion Guide

Case Description

Profile:

Patient G is of Trinidadian descent, female, and born in September 1948. She immigrated to the United States in 1989 and works as a dietary specialist. She lives with her immediate family and has no history of drug, alcohol, or tobacco use. She first presented to a local area clinic in 2005, desiring to transfer her eye care to a facility that was geographically closer to her home. She brought her eye examination records dating from 1998 to 2005. Patient G had been diagnosed with primary open-angle glaucoma in 2001. She was a knowledgeable and compliant patient. She stated that she “never missed” taking her medication. She understood the nature of this asymptomatic, chronic disease and felt it was important to do everything possible to control the disease.

Between 1998 and 2009, the patient had multiple office visits. A summary of relevant examination findings will be presented for four intervals; 1998–2001, 2001–2005, 2005–2007, and 2007–present.

Summary of examination findings: 1998–2001

From 1998 to 2001, the records indicated a history of arthritis, which was treated with Vioxx, and otherwise unremarkable systemic health. Her family history was positive for a sister being treated for glaucoma. Records indicate a corneal scar OS, secondary to a laceration obtained in a car accident in 1970, which resulted in a decrease in visual acuity. During this time (1998–2001), she was followed for ocular hy-

pertension in both eyes. In 2001, after the visual field test, the diagnosis was changed from ocular hypertension to primary open-angle glaucoma, and treatment (OU) was initiated with Travatan. After two months on treatment with Travatan, the patient reported side effects of conjunctival injection and irritation, and medication was changed to Xalatan. Table 1 shows baseline data; Table 2 shows post-diagnosis/treatment data.

Summary of examination findings: 2001–2005 Table 3

From 2001 to 2005, visual acuity and C/D ratio remained stable with no changes. In 2002, the patient requested a change in medications secondary to

a change in her insurance formulary, which no longer covered Xalatan. At this time, her treatment was changed to Lumigan 0.03% SOL (bimatoprost) one drop before bed. In 2005, in addition to the diagnosis of arthritis, the patient was diagnosed with hypercholesterolemia and hypertension. Her medications from this time period included; lisinopril, hydrochlorothiazide, and naproxen.

Summary of examination findings: 2005–2007 Table 4

In 2005, the patient transferred care to our clinical facility. She reported good health with well-controlled systemic hypertension. Current systemic medications were: hydrochlorothiaz-

Table 1
Baseline data before treatment 1998–2001

	OD	OS
Best corrected VA OD +1.25 = -0.50 x 100 OS +1.50 = -2.50 x65	20/20–	20/40-ph ni Secondary to corneal scar
Pupils	ERRL-APD	
TAP 4:00PM, 11/25/98	21 mmHg	19 mmHg
TAP 3:39PM, 3/2/01	22 mmHg	20 mmHg
TAP max no time, 7/17/01	26 mmHg	24 mmHg
Biomicroscope	WNL	Stromal scar
Van Herick Angle	1:1	1:1
Fundus with dilation, C/D	55%	55%
Humphrey Visual Fields (HVF) 24-2 SITA standard-reliable	Scattered, nonspecific defects superior and nasal MD-1.49DB PSD 2.60DB	No defect MD -3.33DB PSD 2.34DB

Table 2
Post diagnosis/treatment 2001

Diagnosis	Primary open-angle glaucoma OU
Initial treatment	Travatan 0.004% SOL (travoprost) one drop before bed, OU
TAP post 2 month Tx @3:08PM, 6/21/01	OD 20 mmHg, OS 20 mmHg
Med change secondary to side effects (Conjunctival injection and irritation)	Xalatan 0.005% SOLN (latanoprost) one drop before bed, OU

ide 25 mg, once per day, lisinopril 10 mg, one tablet per day, and naproxen 500 mg. We referred the patient for a baseline Heidelberg Retina Tomograph (HRT), and pachymetry readings were obtained.

At this time, the point of view of the treating doctor was that the lower the IOP the better. However, a target pressure was not established. This patient expressed her point of view as being very trusting of medical providers; she felt that the medical provider knew what was best for her.

In 2005, during the patient's first visit at our clinical facility, her treatment was changed. Cosopt 2 0.5%, administered twice a day, was added to her list of current ocular medications, which included Lumigan 0.03% SOL, one drop before bed. Her medical history remained stable with no changes in diagnosis or medications. In 2007, the patient reported that her adult son was recently diagnosed and treated for glaucoma. Her C/D remained symmetrical and stable with estimates of 55% OD, OS.

Summary of examination findings: 2007–present Table 5

In December 2007, the diagnosis was reevaluated. The patient was ordered to stop all glaucoma medications for a medication holiday. The optometrist was open to reevaluating all data and information, which included the data gained after the medication holiday. The patient had developed a trusting relationship with the eye care providers and was comfortable taking a medication holiday.

In 2009, atenolol 25 mg, one tablet per day was added to the patient's systemic medications for better control of hypertension. Her C/D remained stable at 55%, symmetrical with healthy rim tissue. A repeat HRT (2008) was performed at the same facility as the first HRT in 2005. Subsequent HRTs performed in 2009 were performed at the current clinic, with new equipment.

Key Concepts

1. Strategy for diagnostic reasoning emphasizing the art of analysis, evaluation, and assessment of thinking⁴
2. Evidence-based medicine: how research can drive and change stan-

**Table 3
Data from 2001–2005**

	OD	OS
Best corrected VA	20/20–	20/40–
Treatment 2001–2002	Xalatan 0.005% SOLN (latanoprost) one drop before bed	Xalatan 0.005% SOLN (latanoprost) one drop before bed
TAP 4:07PM, 11/30/01	18 mmHg	16 mmHg
TAP 3:12PM, 6/3/02	19 mmHg	18 mmHg
Fundus with dilation C/D	55%	55%
Treatment 2002–2005	Lumigan 0.03% SOL (bimatoprost) one drop before bed	Lumigan 0.03% SOL (bimatoprost) one drop before bed
TAP 3:16PM, 10/11/02	17 mmHg	17 mmHg
TAP 6:30PM, 7/12/04	21 mmHg	22 mmHg
TAP 6:12PM, 5/15/05	16 mmHg	15 mmHg
HVF 2002 24-2 SITA standard-reliable	No glaucomatous defects MD –3.28DB PSD 1.33DB	Few scattered defects, no glaucomatous pattern MD –3.01DB PSD 2.46DB

**Table 4
Data from 2005–2007**

	OD	OS
Best corrected visual acuity	20/15	20/40
Treatment (2005) when first presenting to new clinic	Lumigan one drop before bedtime	Lumigan one drop before bedtime
TAP 10:30AM, 5/31/05	22 mmHg	18 mmHg
TAP 6:30PM, 07/12/05	22 mmHg	19 mmHg
Pachymetry (2005)	620 µm	629 µm
HRT (2005)	SD 25 µm, no defects	SD 84 µm, poor quality secondary to corneal scar
HVF (2005) 24-2 SITA fast OD low reliability secondary fixation losses OS reliable	Isolated nasal defect MD –1.10DB PSD 1.59DB	No glaucomatous defects MD –3.92 DB PSD 1.57DB
Fundus with dilation C/D	55% H/V, healthy rim, no heme, moderate depth	55% H/V, healthy rim, no heme, moderate depth
Change in treatment (2005) 11/02/05	Lumigan 0.03% SOL (bimatoprost) one drop before bed and Cosopt 2-0.5% SOLN (dorzolamide-timolol) one drop bid	Lumigan 0.03% SOL (bimatoprost) one drop before bed and Cosopt 2-0.5% SOLN (dorzolamide-timolol) one drop bid
TAP min 7:01PM, 2/13/07	13 mmHg	13 mmHg
TAP 7:09PM, 8/10/07	17 mmHg	17 mmHg
Tap max 6:25PM, 5/13/06	20 mmHg	19 mmHg
Pachymetry (2006)	617 µm	631 µm
HVF (2006) 24-2 SITA fast reliable	No defects MD +0.15 DB PSD 1.25 DB	No defects MD –3.39DB PSD 1.55 DB
HVF (2007) 24-2 SITA fast Borderline reliability secondary to false positives	Scattered defects nasal and inferior ? beginning arcuate defect MD –1.10 DB PSD 2.14 DB	No defects MD –3.33 DB PSD 1.59 DB
Gonioscopy with 3 mirror lens	Open with normal structures, no excessive pigmentation	Open with normal structures, no excessive pigmentation

- dard of care
3. Pathophysiology of glaucoma, integration of basic science concepts in a clinical presentation
 4. Assessment of risk factors, including the concept of steady state and modifiable factors
 5. Reliability, sensitivity, and specificity of diagnostic testing
 6. Treatment, “Do no harm” risk/benefit ratio; role of economic and psychosocial factors.

Learning Objectives

At the conclusion of the case discussion, the participants should be able to:

1. Describe how evidence-based medicine can influence and change standard of care, utilizing recent clinical studies in the analysis of and diagnosis of glaucoma
2. Utilize critical thinking concepts in diagnosis
3. Discuss the importance of the concept of point of view and how it can influence decision-making
4. Describe the role of risk factors, pathophysiology, diagnostic testing, and pharmacology in the diagnosis and treatment of glaucoma and management of ocular hypertension.

Discussion Questions

The depth of the discussion questions may vary according to the academic level of the student. All discussion questions should be applied to primary open-angle glaucoma.

A. Concepts, Knowledge, Facts Required for Critical Review of the Case

1. What concepts are involved in critical thinking? Discuss analysis, evaluation, and reflection.
2. What tissue and structures are involved in primary open-angle glaucoma? What happens at a cellular level? Relate the disease process at the tissue or cellular level to the signs and symptoms of the disease.
3. What are the risk factors for glaucoma? How are the risk factors used in the management of glaucoma?
4. What diagnostic tests, according to standard of care, are

Table 5
Data from 2007–present (post medication holiday)

	OD	OS
Fundus with dilation C/D	55% H/V, healthy rim, no heme, moderate depth	55% H/V, healthy rim, no heme, moderate depth
TAP 6:45PM 1 month post holiday, 12/18/07	20 mmHg	19 mmHg
TAP 7:30PM 6 month post holiday, 6/10/08	21 mmHg	22 mmHg
HRT (2008)	SD 34 μ m, no defects	SD 87 μ m, no defects
TAP 7:49PM 13 month post holiday, 1/20/09	22 mmHg	22 mmHg
TAP 9:15AM 14 month post holiday, 2/3/09	28 mmHg	28 mmHg
TAP 9:35AM 15 month post holiday, 2/10/09	28 mmHg	26 mmHg
TAP 7:12PM 16 month post holiday, 4/7/09	16 mmHg	18 mmHg
HRT (2/2009), new equipment	SD 19 μ m one borderline defect, temporal	Poor image
HRT (5/2009), new equipment	SD 15 μ m no defects	Poor image
HVF (2009) 24-2 SITA fast reliable	No defects MD -0.83 PSD1.30 DB	No defects MD -3.20 DB PSD1.72 DB

needed to follow a patient with ocular hypertension?

5. Summarize the important clinical studies used in the management of ocular hypertensive patients.
 6. What medications are commonly used in the treatment of glaucoma? What are the modes of action, side effects (short and long term), costs, etc.
- B. Critical Analysis: Purpose, Question, Information, and Inferences
1. What is your goal/objective in caring for this patient?
 2. What evidence/information do you need to evaluate this case?
 3. Is there sufficient evidence to diagnose glaucoma?
 4. At each interval, what evidence supports/negates the diagnosis of glaucoma?
 5. If the diagnosis is ocular hypertension, what evidence supports/negates the decision to treat?
 6. How should we interpret the data? Discuss the inferences/con-

clusions that can be made from the data, including the evaluation of the reliability of evidence such as HVF and HRT.

7. Discuss the inference/conclusions that can be made from the relevant clinical studies.
- C. Critically Assessing: Points of View and Assumptions
1. What assumptions are made in this case?
 2. How do the assumptions impact the case?
 3. What information can be obtained to justify the assumptions?
 4. What are the different points of view involved in the case?
 5. How do the different points of view impact the case?
- D. Critically Assessing: Implications
1. Discuss the implications of treating or not treating. Consider cost, risk/benefit ratio, psychosocial issues, side effects, time, evidence, points of view, etc.
 2. Discuss the psychosocial issues

and implications of these issues. Consider doctor/patient relationship, compliance, anxiety of patient and doctor, etc.

3. Discuss implications of under-versus over-treatment.
4. Discuss the implications of systemic medications on intraocular pressure.
5. Discuss the psychological implications of changing or removing a diagnosis.

Recommended Reading

1. Paul R, Elder L. *Critical Thinking, Tools for Taking Charge of Your Learning and Your Life*. Upper Saddle River, New Jersey, Pearson Prentice Hall; 2006.
2. Facione NC, Facione PA. *Critical Thinking and Clinical Reasoning in the Health Science*. Millbrae: California, Academic Press; 2008.
3. Nosich G. *Learning to Think Things Through*. Upper Saddle River, New Jersey, Pearson Prentice Hall ; 2009

Educator's Guide

The educator's guide contains the information needed to discuss the case. Individual educators should tailor the information to a level appropriate for students in the first and second year of optometric education.

Background

The American Academy of Ophthalmology defines primary open-angle glaucoma (POAG) as a chronic, progressive disease that most often presents with characteristic optic nerve damage, nerve fiber layer defects, and subsequent visual field loss. It occurs primarily in adults and is generally bilateral but not always symmetrical in presentation. The majority of people with POAG have elevated intraocular pressures.⁵ There is currently no single agreed-upon standard reference for identifying POAG.⁶ Ocular hypertension is defined as an abnormal level of IOP (IOP >21mm Hg) with normal visual fields and nerve head.⁷ The Baltimore Eye Study estimates that 8% of adults over the age of 40 have ocular hypertension.⁸ The management of patients with ocular hypertension is complicated by the general agreement among eye care pro-

fessionals that a "safe" or "normal" level of intraocular pressure differs among patients. Therefore, potential risk factors are an important component in the care of ocular hypertensive patients.²

Strategy for Critical Thinking

Introductory information on critical thinking is presented. To expand your knowledge of critical thinking concepts, refer to the list of recommended reading. The recommended readings are appropriate for both students and faculty. Table 6 provides a summary of basic information for critical thinking and the parts of analysis.

Critical thinking provides a guide or strategy for accurate and efficient clinical judgment. Critical thinking involves thinking about thinking, with the aim of improving thinking.⁴ Critical thinking utilizes analysis, evaluation, and reflection of thinking.⁴ Analysis focuses on the parts of thinking: its purpose, question, information, inferences, assumptions, concepts, implications, and point of view.⁴ Evaluation indicates assessing the clarity, accuracy, precision, relevance, depth, and breadth of thinking.⁴ Reflection involves thinking about thinking to build on its strengths while reducing weaknesses.⁴ The teaching and utilization of critical thinking skills

within the framework of a case report will aid students in achieving the skills of an ideal thinker.

One characteristic of an ideal thinker is the ability to recognize and differentiate information, inferences, assumptions, and points of view.⁹ Inferences are made to make sense of information and are influenced by assumptions and point of view.⁹ Inferences and assumptions can be accurate or inaccurate, justifiable or unjustifiable, or logical or illogical.⁹ Good clinical judgment often involves making inferences based on assumptions. For students to develop good clinical judgment they must develop the ability to recognize assumptions and determine the quality of the assumption.

Pathophysiology of POAG

Glaucoma is a multifactorial optic neuropathy that ultimately affects the individual axons of the optic nerve. We know that obstruction to axoplasmic flow is the culprit in the pathogenesis of glaucomatous optic atrophy through a process of programmed cell death called apoptosis. However, we still do not know if this cascade of events is initiated by mechanical or vascular factors or by a mechanism that we have not yet discovered.

Table 6⁴
Parts of Analysis

Parts of Analysis	Definition
Purpose	Your purpose is your goal, your objective, what you are trying to accomplish.
Question	The question(s) lays out the problem or issue and guides thinking.
Information	Information includes the facts, data, evidence, or experiences we use to figure things out.
Inference	Inferences are interpretations or conclusions you reach. Inferring is what the mind does to figure something out.
Assumptions	Assumptions are beliefs you take for granted or presuppose.
Concepts	Concepts are ideas, theories, laws principles, or hypotheses we use in thinking to make sense of things.
Point of View	Point of view is literally the place from which you view something.
Implications	Implications are the things that might happen if you decide to do something.

According to the mechanical compression theory, elevated IOP causes a backward bowing of the lamina cribrosa, kinking the axons as they exit through the lamina pores. This may lead to focal ischemia, deprive the axons of neurotrophins, or interfere with axoplasmic flow, triggering cell death.¹⁰

According to vascular theories, death is triggered by ischemia, whether induced by elevated IOP or as a primary insult. Reduced blood flow to the optic nerve starves the cells of oxygen and nutrients. This reduced flow may be associated with systemic factors, such as migraine headaches, cardiovascular disease, diabetes, systemic hypertension, and systemic hypotension.¹⁰

There is a genetic theory that cell death is triggered by genetic predisposition. Following the death of individual axons, substances may be released into the environment causing a secondary triggering of apoptosis in neighboring cells, including glutamate (a neurotransmitter that may cause excitotoxicity), calcium, nitric oxide, and free radicals. Some glaucoma patients exhibit elevated levels of the neurotransmitter glutamate within the vitreous. Ganglion cells contain protein receptors that, when activated by glutamate, increase intracellular calcium to toxic levels, killing the cells.¹¹

It may be that all of these theories are involved to some degree or that there is more than one mechanism of optic atrophy that depends on each individual's particular susceptibility.

Research has suggested that the vascular theory may be the predominant factor in patients who develop glaucoma in spite of a low IOP, whereas the mechanical theory is more prevalent in patients with higher IOP.¹⁰

Risk Factors for POAG

When discussing risk factors for POAG, it is important to note that associations do not imply causation. They do, however, help us in identifying high-risk populations for whom close observation and routine screenings would be most beneficial. There is strong evidence supporting age, race, family history, high myopia, and intraocular pressure as risk factors.¹² In fact, the evidence for intraocular pressure as a potential risk factor for glaucoma is so strong that it is considered one of the potential causes as well as a risk. There is moderate evi-

dence for large cup-to-disc ratio, optic disc hemorrhage, and diabetes posing increased risk for glaucoma. There is slightly weaker evidence showing an association between glaucoma and systemic conditions, such as increased systolic blood pressure, migraine headaches, hypothyroidism, sleep apnea, and autoimmune disease.¹⁰ It is also important to note that some systemic medications may increase a patient's risk for development of glaucoma. No environmental, infectious, or social risk factors have been identified. Recognition of a patient's risk factors for glaucoma can lead to earlier detection and better management.

Risk Assessment

It is important to incorporate evidence-based medicine into the decision-making processes when evaluating a patient's risk for development of glaucoma. There are several ongoing clinical trials that provide evidence for the risk factors that we have discussed above, as well as for the importance of early intervention and treatment of those with high risk for the development of glaucoma.¹³ The Ocular Hypertension Treatment Study evaluated the effect of treatment vs. no treatment in ocular hypertensive patients and found that

lowering IOP with medical therapy reduces by half the rate of conversion to open-angle glaucoma in high risk individuals.² This study also supports age, IOP, cup-to-disc ratio, and central corneal thickness as baseline factors that may predict the onset of glaucoma. The Early Manifest Glaucoma Treatment Study examined the effects of treatment vs. no treatment in newly diagnosed POAG patients. The study demonstrated that lowering IOP with medical therapy and argon laser trabeculoplasty inhibits progression of optic disc and visual field damage by 50%.¹⁴

Analysis of long-term, population-based studies of patients with high IOP and glaucoma reveals that treatment may reduce the risk of progression from untreated high IOP to blindness by a range of 1.2 percent to 8.1 percent over 15 years.¹⁵ It has also been determined that between 12 and 83 patients with high IOP require treatment to prevent one patient from progressing to unilateral blindness over a 15-year period.¹⁵ A brief summary of relevant clinical trials is listed in Table 7.

Results from these studies can be used in conjunction with a thorough case history and examination findings to assess a patient's risk for development of

**Table 7
Clinical Trials**

Study	Description	Summary
Collaborative Normal Tension Glaucoma Study	Compared topical drug and/or surgical treatment to no treatment for normal-tension glaucoma patients	Over 10 years, it was found that a 30% IOP reduction cut glaucoma progression by 50%
Advanced Glaucoma Intervention Study	Compared effect of trabecular surgery type on black and white glaucoma patients	After 10 years, IOP was lower for both racial groups (but the best surgery type was race-specific)
Collaborative Initial Glaucoma Treatment Study	Compared trabecular surgery first versus topical drug treatment first for glaucoma patients	Both groups had equal IOP reductions after 5 years
Ocular Hypertensive Treatment Study	Compared topical drug treatment versus no treatment for ocular hypertensives	Treatment reduced progression to glaucoma by over 50% and thin central corneal thickness increased risk of glaucoma
Early Manifest Glaucoma Trial	Compared effects of argon laser trabeculoplasty plus betaxolol treatment versus no treatment over an average period of 6 years	Argon laser trabeculoplasty plus betaxolol reduced progression of glaucoma by 50% more than progression without treatment

glaucoma and support or refute a decision to initiate treatment. Multicenter data from the Ocular Hypertension Treatment Study and the Early Manifest Glaucoma Treatment Study allowed for the development of the glaucoma risk calculator that may be downloaded and used to evaluate a patient's risk for development of glaucoma.

Testing used to aid in the diagnosis of POAG

In this case, the Heidelberg Retina Tomograph (HRT) was used to evaluate the structure of the optic nerve and nerve fiber layer. The HRT is a laser imaging technique that uses confocal scanning laser ophthalmoscopy to produce pseudo-three dimensional images of the optic nerve head, as well as numerical measurements (e.g. rim area, rim volume, cup shape measure, height variation contour, mean retinal thickness, etc.).¹⁶ These numerical measurements can be used to document the optic nerve head and monitor changes in measured parameters. The HRT also performs a Moorfields Regression Analysis, which is a statistical tool allowing us to compare each patient's parameters to those of a normative database (meaning averaged values over a large group of normal patients who do not have glaucoma). From this, the examiner can statistically predict the likelihood of the individual patient having optic nerve changes suggestive of glaucoma.¹⁶

Automated visual field testing with the Humphrey field analyzer is the most frequently used psychophysical test for measuring and monitoring the function of the optic nerve. Factors such as patient selection, test selection, test reliability, and proper interpretation of test results are important when using visual field testing to diagnose and manage glaucoma.¹¹

Although most patients are capable of performing visual field tests, modifications to testing technique must be made to compensate for patients with physical or mental limitations (e.g. elevation and stabilization of the head, frequent rests, and test-shortening strategies). It is important that the clinician recognize when such modifications are necessary in order to assure reliable, consistent automated field results.¹¹

The Humphrey visual field analyzer offers a battery of test strategies from

which the clinician may choose. Table 8 shows the most commonly used tests.

In selecting which test strategy to use, the clinician should consider certain factors, such as the patient's physical or mental limitations, first-time test-taker vs. experienced test-taker, and level of suspicion/risk for glaucoma. The testing strategy should be kept as consistent as possible when monitoring a patient for visual field changes, so that any change in the field is attributable to the disease process rather than a change in the testing condition. Close attention should be given to reliability indices (fixation losses, gaze tracking, false-negatives, false-positives) as well as possible artifacts, pseudodeficits, and defects from other diseases.¹¹

Field defects commonly found in early glaucoma include nasal steps, paracentral scotomas, visual field loss in the upper hemifield that is different com-

pared with the lower hemifield, and defects within the arcuate bundle. As the disease progresses, further loss of axons causes these defects to coalesce. In end-stage disease, only a central and/or temporal island remains.¹² The determination of whether or not a defect is glaucomatous is not always straightforward and can be challenging. Several algorithms are available, either by computerized printout or computed by the practitioner. These algorithms can be complex and are beyond the scope of this paper. However, there is extensive literature readily available to aid the clinician in the analysis/interpretation of visual field defects.^{11,12,16}

Pharmacology of Medical Treatment of POAG

Medical treatment is usually the first line of treatment for POAG and includes the use of several classes of topical agents. All act by reducing intraocu-

**Table 8:
Test Strategies for the Humphrey Visual Field Analyzer**

Name of Test	Points Tested	Comments
Full Field 120 (F120)	120 points over full field	Good sensitivity, fair specificity; useful as practice test
Full Threshold 30-2	76 points in central 30 degrees	Gold standard; longest test
Full Threshold 24-2	54 points in central 24 degrees (eliminates all edge points from 30-2 except for 2 most nasal points on horizontal meridian)	Reduces test time by 10%-20%; comparable sensitivity and specificity to the 30-2 test
Full Threshold 10-2	68 points in central 10 degrees	Preferred test in end-stage glaucoma or when fixation is threatened
SITA Standard 30-2 or 24-2	76 points in central 30 or 24 degrees	Determination of threshold is reached with fewer questions asked; reduces test time by 50% of the time it takes for a full threshold; Same accuracy as full threshold
SITA Fast 30-2 or 24-2	76 points in central 30 or 24 degrees	Requires a lower level of confidence to stop testing at a given point; reduces test time by 25% of the time it takes for a full threshold; less accurate than SITA Standard
FASTPAC	variable	Can be used in conjunction with 10-2 or 24-2 test patterns to reduce test time by as much as 40%; less precise than full threshold; scotomas may be underestimated

lar pressure. The main classes of topical agents used in the treatment of POAG include prostaglandin analogues, topical beta-blockers, alpha agonists, and carbonic anhydrase inhibitors (Table 9).¹⁰ Two topical agents may be combined if one agent is ineffective.

Discussion

This section will focus on key elements to stimulate discussion and is not inclusive of all possible discussion points. This discussion focuses on the use of critical thinking, while actual classroom discussion should include more in-depth integration of knowledge, current literature, and expertise.

With regard to the time period from 1998 to 2001, several important questions should be formulated and discussed. What is the differential diagnosis? What information is needed to support or rule out the diagnosis? What is the validity of the information? What are this patient's risk factors for glaucoma?

Patients with POAG present to clinicians at different stages of the disease. The earliest stages are asymptomatic with undetectable acceleration of apoptosis, ganglion cell death and retinal nerve fiber layer changes.⁶ Later stages are still asymptomatic but with detectable nerve fiber layer changes. As the disease progresses, the symptom of visual field loss initially may or may not impact function.⁶ Therefore, the man-

agement and analysis of risk factors becomes a key element in the diagnosis and management of POAG. What evidence (risk factors) puts the patient at risk for glaucoma? Are all risk factors equal? What clinical studies identify risk factors for primary open-angle glaucoma? How do clinicians evaluate clinical studies? From 1998 to 2001, information about IOP, family history, visual fields, ethnicity, and optic nerve head evaluation is needed to evaluate the patient's risk for glaucoma.

The evidence (risk factors) that puts this patient at risk is: positive family history (sister), elevated IOP, age, and visual field results. Data is random information unless it is analyzed. Analysis of information is guided by our knowledge base and experience and leads to reasonable inferences and conclusions.

Review of the literature reveals many different reports of a statistical mean IOP. Leydhecker's investigation of 10,000 individuals without glaucoma indicates an average IOP of 15.5 mmHg +/- 2.57 mmHg.¹¹ Additionally, "normal" levels of IOP differ among patients. Therefore, it is difficult to conclusively determine if the patient's IOP is outside the normal range for that particular patient. Referring to the definition of ocular hypertension, the patient's pressure readings of high teens, low twenties does put her in the borderline ocular hypertensive range. One high reading of 26 mmHg, 24mmHg

is recorded. It is impossible to analyze the significance of this reading since the time of the reading was not recorded. Based only on IOP readings during this time period, the diagnosis of ocular hypertension is reasonable to consider.

Most clinicians at the time assumed that the measured IOP accurately reflected the actual IOP. Information was published in the 1990s dealing with the accuracy of applanation tonometry.⁶ There are two hypotheses to address why this assumption was not questioned: most clinicians at that time were conservative about incorporating new information into clinical practice, or the clinician was unaware of the findings in the literature. The record indicates that, based on the IOP, the patient was diagnosed as ocular hypertensive.

In 2001, the patient was 53 years old. There is significant evidence that with each decade of life there is an increased risk of progression in patients with ocular hypertension to glaucoma.¹⁷ The Beaver Dam Eye Study reported the prevalence of open-angle glaucoma in people aged 43 to 54 years was 0.9%, while it was significantly greater, 4.7%, in individuals 75 years and older.¹⁸

In 2001, family history was a known risk factor for glaucoma.¹⁸ Patients with a first-degree relative who had the disease were estimated to be 4-8 times more likely to develop the disease.¹⁹ Family history may reflect genetic simi-

**Table 9:
Medical Treatment for POAG**

Class	Mechanism of Action	Examples	Side Effects
Prostaglandin analogues	Increase aqueous outflow	Xalatan, Travatan, Lumigan	Changes in eye color and eyelid skin, stinging, blurred vision, eye redness, itching, burning
Beta-blockers	Decrease aqueous production	Timolol, Timoptic XE, Betoptic, Carteolol	Low blood pressure, reduced pulse rate, fatigue, shortness of breath; rarely, reduced libido, depression
Alpha agonists	Decrease aqueous production	lopidine, Alphagan (brimonidine)	Burning or stinging, fatigue, headache, drowsiness, dry mouth and nose, relatively higher likelihood of allergic reaction.
Carbonic anhydrase inhibitors	Decrease aqueous production	Trusopt (dorzolamide), Azopt (brinzolamide), Diamox, Neptazane	Stinging, burning, eye discomfort; in pill form, tingling hands and feet, stomach upset, memory problems, depression, frequent urination
Combination	Decrease aqueous production	Cosopt (timolol/dorzolamide), Combigan (brimonidine/timolol)	Side effects of combined medications may include any of the side effects of the drug types they contain

larities related to the disease process, IOP, optic nerve head anatomy, shared previous exposure, or access to health care.²⁰ Therefore, the patient's self-reported information that her sister was being treated for glaucoma, increased her risk of the disease.

As clinicians, we routinely ask patients to report on their family history. From this information, inferences and conclusions about the patient's risk of disease are formulated by the clinician. The inferences and conclusions are based on the assumption that the patient's report and the diagnosis of a family member's disease is accurate. One goal of critical thinking is to decide what to believe in a given context, with available evidence, knowledge, and criteria.²¹ What evidence can be elicited to justify the assumptions? Due to time, privacy, and practical limitations, most clinicians are unable to review evidence related to accuracy of information about family history. Therefore, it is important that each clinician recognize the assumptions made and use his or her best judgment to evaluate information and justify assumptions.

The initial visual field was taken one month before the diagnosis of glaucoma. Analysis of the visual field does indicate a cluster of defective points, which could indicate a nasal defect in the right eye. The mean deviation in the left eye was decreased. Questions to discuss: Is a one-time visual field defect a reliable piece of evidence? What can we infer or conclude from this piece of data? What is the significance of the mean deviation and does it impact the case? The reliability and reproducibility of the evidence must be explored before any conclusions can be drawn. Most experienced clinicians would not draw conclusions from one visual field result.²

In 2001, after the visual field, the clinician involved in the patient's care concluded that the evidence was sufficient to support the diagnosis of glaucoma, and treatment was initiated. Is this a reasonable conclusion? What other evidence is available to analyze? What is the doctor's hypothesized points of view on diagnosing and treating glaucoma? What evidence is missing?

The diagnosis of primary open-angle glaucoma is based on ruling out secondary causes including the filtration angle

being anatomically normal and observable by gonioscopy. The initial angle evaluation reported was from the Van Herick technique. A definitive test, such as gonioscopy, either was not performed or not recorded at that time. Therefore, the diagnosis of primary open-angle glaucoma was inferred based on the assumption that if the angle by Van Herick is open, the anatomical angle is actually open and normal in structure. Is this assumption justifiable?

The patient's ethnicity was not recorded on any exam form from 1998 to 2005. Since we lack specific information, we can infer that ethnicity was not elicited and not evaluated as a risk factor. This is based on the assumption that if it is not recorded, it was not elicited.

The C/D ratio was initially recorded as 55% OD, OS. From this information, we can infer that the cups were symmetrical with no vertical elongation. Other common glaucomatous signs were not mentioned in the record. It is impossible to conclude if the clinician did not look for other signs of glaucoma or if that information was just not recorded. The importance of accurate record-keeping and recording negative findings should be emphasized. As with ethnicity, in the absence of specific information, clinicians are inclined to make inferences based on assumptions. Is it reasonable to assume that saucerization, disc heme, peripapillary atrophy, abnormalities of blood vessels and excessive depth of cup were not present? Is a C/D ratio of 55% a risk factor?

In 2001, treatment was initiated with the prostaglandin analogue anti-glaucoma medication, Travatan, followed by Xalatan and then Lumigan. The changes in medications were precipitated by side effects from Travatan and a change in the patient's formulary, which no longer allowed for coverage of Xalatan. These prostaglandin analogue anti-glaucoma drugs work by increasing uveal scleral outflow. The anticipated drop in IOP from this class of drugs is an average of 30%.²² Over the next 4 years, the IOP never consistently reached the desired outcome from the medication. Is this finding significant? Is it an indication to change or increase the medication? Compliance to treatment must always be investigated when desired medication results are not achieved. The patient reported excel-

lent compliance with medication. We are assuming the patient is being honest. What evidence can we ask for to justify the assumption?

Over this time period, C/D ratio and visual fields remained stable with no glaucomatous progression.

In 2005, the patient transferred care. New information from pachymetry and Heidelberg Retinal Tomography were added to the database. The pachymetry demonstrated thick corneas of 620 nm and 629 nm. What is the relevance and significance of this information? Corneal thickness can influence the accuracy of IOP as measured by applanation tonometry.²³ Why is the corneal thickness so important in this case? Thicker-than-average corneas may result in an overestimation of IOP. The Ocular Hypertensive Treatment Study demonstrates an increased risk for ocular hypertensive patients with thin corneas. The corneal thickness is a critical piece of evidence. Could any other conditions such as edema, scars, etc. influence the pachymetry measurement? Is the reading reliable? Is the reading reproducible? The corneal thicknesses were confirmed with repeat measurements the following year.

The initial diagnosis was based on the assumption that the measured IOP accurately reflected the actual IOP. This assumption would have a large impact over the course of 10 years.

Results from the HRT (2005) provide an additional piece of information. The Moorfields Regression Analysis indicates normal rim tissue as compared to a normative database. What conclusions can be drawn from this information? Reports of sensitivity and specificity of HRT parameters in detecting glaucoma are good.²⁰ The HRT has more longitudinal patient data than other imaging devices.²⁰ As a result, the HRT's role in determining disease progression is well-established.²⁰ However, analysis of the information must consider test susceptibility to media opacities, subjectivity of setting the contour lines, and defining the normative database and cannot be used as a conclusive diagnostic tool at this time.

Analysis of HVF (2005) indicates an isolated nasal defect in the right eye and no defects in the left eye. A nasal defect in the right eye was first reported in the

initial baseline HVF, however this defect was not repeated in 2001. The visual field from 2005 demonstrated low reliability, secondary to fixation losses. What conclusions, if any, can we draw from this information? Would additional information from other types of visual field testing, such as frequency-doubling technology or short wavelength automated perimetry be beneficial or influence decision-making? What is the role of visual field testing? Is it detection, progression, or both?

The C/D ratio remains stable, with healthy rim tissue, no disc heme and moderate depth. No glaucomatous progression is reported. What is the best way to monitor the status of the optic nerve?

The point of view of the treating doctor at this time (2005) was that for patients who are diagnosed with glaucoma, the lower the IOP the better. As a result of this point of view, the treatment was changed to include Cosopt 2 in an effort to further lower IOP. The merits and limits of establishing a target IOP and the aggressiveness of the treatment can be discussed. The patient was willing to add the additional medication and reported good compliance with the new treatment plan. Patients often present with different points of view on medical care. Some patients view the doctor as an authoritarian and will not question the diagnosis or treatment, while others want to be considered a partner. This patient's point of view was of the doctor as an authoritarian, and she willingly complied with the treatment.

In 2007, clinicians came to the conclusion that the diagnosis of glaucoma needed to be reevaluated, and a medication holiday (temporary discontinuation of glaucoma medications) was considered. Clinicians now had the benefit of information from the Ocular Hypertension Treatment Study and analysis of data over time. This represented the ability to review multiple IOP measurements, HVF, HRT, and fundus evaluations. The HVF from 2007 before the medication holiday indicated a questionable arcuate scotoma. Before deciding to initiate the holiday, several important questions need to be evaluated. Does the evidence still support the diagnosis of glaucoma? What evidence supports the diagnosis of ocular hypertension? If the diagnosis is ocular hypertension, should the patient

be treated? Does the visual field need to be repeated? A clinician's point of view can be influenced by past experiences, knowledge base, new information, and new technology. Willingness to reevaluate represents open-mindedness, flexibility of thinking, skills in habitually questioning, and willingness to reconsider and reflect on thinking. This includes questioning previous diagnoses made by the same clinician or another clinician.

In 2007, evidence supporting the diagnosis of glaucoma is age and positive family history, sister, and son. Is family history a fact or an assumption? What information on family history can we gather from the literature? Could the patient's sister and brother have inherited thick corneas?

Evidence not supporting the diagnosis of glaucoma is lower IOP due to the influence of thick corneas, no repeatable glaucomatous defects on HVF, C/D ratio stable (symmetrical with no glaucomatous progression or defects reported), no defects on HRT and thick corneas. Is the lack of progression the result of lack of disease or the result of effective treatment?

The role of ethnicity is difficult to evaluate. The ethnicity of the people of Trinidad was influenced by many different cultures.²⁴ Because of this fusion of cultures, it is impossible to draw any accurate conclusions on the role of ethnicity as a risk factor.

A medication holiday was initiated in 2007 to gain additional information. The specific length of time for the medication holiday was not initially established. The plan was to monitor the patient closely and evaluate her response to the discontinuation of medication. Post-holiday IOPs were consistently stable 3 mmHg to 4 mmHg above treatment IOP with diurnal variation 6 mmHg to 7 mmHg higher. Is the diurnal variation significant?

The psychological implications of a change in diagnosis and discontinuation of treatment needs to be explored. This patient always reported good compliance with treatment, demonstrated trust and respect for her doctors, and had been educated about the potential sight-threatening prognosis of untreated glaucoma. Initially, the patient was educated about the use of new tests, such as pachymetry, and new clinical

studies, which necessitated a reevaluation of her diagnosis. She was compliant with discontinuing medications for the first few months. However, after 6 months she became very concerned about the possible removal of her diagnosis and the continual discontinuation of the medications. What are the psychological implications of a change in diagnosis? What are the psychological implications of discontinuing glaucoma medications?

After the patient was off medications for one year, HRT (2/2009) demonstrated one questionable area. Is this a true potential defect, or does this represent the variability of the test with a different examiner on a new piece of equipment? Repeat testing (5/2009) indicated no defects. After the patient was off medications for one year, HVF indicated a reliable test-taker and no defects. The question remains: Is this glaucoma? Is this ocular hypertension? Does this patient need treatment?

Conclusion

Critical thinking is the strategy that allows clinicians to accurately and efficiently use their knowledge base and experiences in clinical reasoning. Acting without adequate analysis, failure to recognize assumptions and points of view, failure to evaluate implications, and unwillingness to reflect and reevaluate behavior are characteristics detrimental to a clinician's ability to provide the highest level of care. This case demonstrates the challenges related to diagnosis and treatment. There are no clearcut conclusions to this case, and, therefore, it provides an excellent stimulus for debate. In the future, new clinical trials and technology, such as risk management calculators and other optical imaging devices, may provide additional useful information. Developing good critical thinking skills will last a student a lifetime. It will allow students the ability to judge new information, incorporate relevant information into clinical practice, and provide the highest level of patient care.

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The original HVF, HRT, and disc photos are available by contacting the corresponding author at deniala@neco.edu.